TABLE 1 Baseline Characteristics, Time Delays, and Outcomes

Baseline clinical characteristics	
Female	37 (44)
Anterior stroke	82 (97)
Age, yrs	64.8 ± 13.8
Diabetes mellitus	25 (30)
History of hypertension	63 (75)
Clinical evidence of atherosclerosis	37 (44)
Atrial fibrillation (any type, any time)	34 (40)
History of stroke or TIA	9 (11)
Admission NIHSS	18.0 ± 4.1 (median 18, range 6-27)
Time delays: median values, min (IQR: 25-75)	
Stroke onset to CT	90 (55-145)
CT to sheath insertion	64 (24-89)
Sheath insertion to recanalization	53 (41-70)
Stroke onset to sheath insertion	165 (95-260)
Stroke onset to recanalization	236 (202-342)
Procedural data	
Intubation/general anesthesia use	24 (29)
Heparin dose, units	3,570 ± 3,800 (median 2500)
Angiographic and clinical outcomes	
Recanalization rate (TICI 2a/2b/3 flow)	62 (74)
Good neurological outcome at 90 days (mRS ≤2)	35 (42)
90-day mRS among early presenters (stroke onset to sheath insertion time <3 h)	3.15 ± 2.20 (median 2)*
90-day mRS among late presenters (stroke onset to sheath insertion time >3 h)	3.81 ± 2.11 (median 4)*
90-day mortality	27 (32)
Symptomatic intracranial hemorrhage at 7 days (%)	12 (14)

Values are n (%) or mean ± SD, unless otherwise noted. *p = 0.160.

CT = computed tomography; IQR = interquartile range; mRS = modified Rankin scale; NIHSS = National Institutes of Health Stroke Score; TIA = transient ischemic attack; TICI = Thrombolysis In Cerebral Infarction.

class IA indication). In contrast, interventional cardiology services for acute myocardial infarction are available on a 24/7 basis in almost all European and North American countries, and are becoming more available on other continents. Thus, in places where neuroradiology services are not available, the involvement of interventional cardiologists may be a reasonable option.

Direct mechanical thrombectomy performed by a cardiologist may be considered a treatment option for acute stroke in centers where no neurointerventional services exist. Cardiology centers are able to achieve short CT to catheter laboratory times due to their experience in primary angioplasty for acute myocardial infarction. Outcomes are comparable to endovascular interventions performed in neuroradiology centers.

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Net Clinical Benefit for Oral Anticoagulation, Aspirin, or No Therapy in Nonvalvular Atrial Fibrillation Patients With 1 Additional Risk Factor of the CHA_2DS_2-VASc Score (Beyond Sex)

Whether to anticoagulate patients with atrial fibrillation (AF) and 1 stroke risk factor (i.e., CHA_2DS_2-VASc [congestive heart failure, hypertension, age

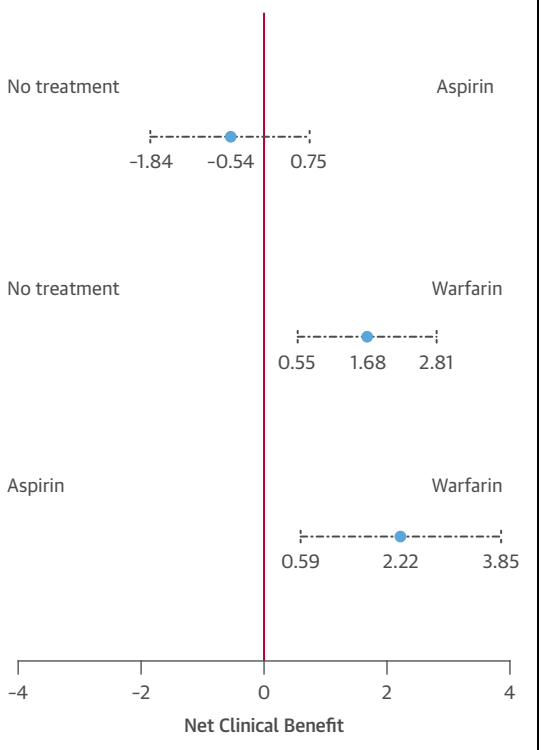
>75 years, diabetes mellitus, stroke, vascular disease, age 65-75 years, and female sex] score = 1 in men, or 2 in women) is controversial, but many studies report ischemic stroke rates of >1.5% per year, even with 1 stroke risk factor (1). We estimated the net clinical benefit (NCB) of aspirin or warfarin compared with no antithrombotic therapy among such patients on the basis of a nationwide Danish cohort, with incident AF diagnosed between 1998 and 2012 (2). Men with a CHA_2DS_2-VASc score ≠ 1 and women with score ≠ 2 at the date of discharge after diagnosis were excluded, as were patients who initiated non-vitamin K antagonist (VKA) oral anticoagulants (OACs), phenprocoumon, or who had warfarin or aspirin prescriptions between 4 and 12 months and before the date of AF diagnosis. Others were stratified according to: 1) no treatment, if there was no prescription for warfarin or aspirin within 1 year; 2) aspirin; or 3) warfarin prescriptions within 4 months.

Endpoint rates were calculated as events per 100 person-years. NCB was calculated for aspirin or warfarin versus no treatment, and for aspirin versus warfarin under intention-to-treat as a weighted sum of rate differences $\Delta R = Rate_{not\ treated} - Rate_{treated}$: $NCB = w_1 \times \Delta R_{ischemic\ stroke} + w_2 \times \Delta R_{ICH} + w_3 \times \Delta R_{major\ bleeding} + w_4 \times \Delta R_{MI}$. When contrasting aspirin and warfarin, the rate difference was given as $\Delta R = Rate_{aspirin} - Rate_{warfarin}$. Positive NCB favored treatment (i.e., warfarin compared with aspirin, or no treatment, respectively). Weights in hazard ratios (HRs) were estimated from the entire Danish AF cohort by Cox proportional hazards models (3), on the basis of 1-year follow-up. NCB with 95% confidence intervals (CIs) were calculated from rate differences and SEs using Poisson regression.

NCBs for 1 follow-up are presented in **Figure 1**, on the basis of weights: $w_1 = 1$, $w_2 = 2.44$, $w_3 = 0.67$, and $w_4 = 0.86$. NCBs were positive for warfarin versus no treatment (HR: 1.68; 95% CI: 0.55 to 2.81) and for warfarin versus aspirin (HR: 2.22; 95% CI: 0.59 to 3.85). The NCB for aspirin versus no treatment was negative (HR: -0.54; 95% CI: -1.84 to 0.75).

This analysis supports a positive advantage for stroke prevention with OACs compared with no therapy or with aspirin among patients with AF who have a single stroke risk factor other than a sex factor (i.e., CHA_2DS_2-VASc score = 1 in men or 2 in women). The analysis by Friberg et al. (4) excluded patients who received OACs during follow-up, which introduced bias away from the null hypothesis that patients with 1 additional risk factor benefit from OAC treatment.

FIGURE 1 Net Clinical Benefit for Anticoagulation, Aspirin, or No Therapy



Net clinical benefit analyses for 1 year of follow-up, for aspirin versus no treatment, warfarin versus no treatment, and for warfarin versus aspirin.

Ischemic stroke rates among such AF patients have varied in reports from different cohorts, reflecting the populations and settings from which the data are derived (1,5). However, clinical risk status changes over time, and hospitalized AF patients face greater mortality and morbidity, whereas OAC reduces stroke and all-cause mortality compared with placebo and/or control drugs. Finally, NCB may be greater with the non-VKA OACs than warfarin, because of their better efficacy and safety compared with the VKAs in clinical trials.

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Stent Type and Risk of Late Cerebral Events After Carotid Artery Stenting

We read with great interest the recent paper by Gensicke et al. (1), which reported a 3-fold increased risk of recurrence of stroke or transient ischemic attack at 6 months in symptomatic patients treated with carotid artery stenting (CAS) and who showed new silent ischemic cerebral lesions on diffusion-weighted magnetic resonance imaging after the endovascular procedure. This observation did not apply to patients treated with carotid endoatherectomy (CEA). The investigators discussed the possible role of vulnerable plaques and the potential benefits of longer and more aggressive antiplatelet therapy. The study provided highly valuable new scientific evidence that might improve the current clinical outcomes of CAS and generated a hypothesis that deserves intensive investigation. However, the previously published ICSS (International Carotid Stenting Study) data provided little information on the kind of stent that was used (2). With cerebral protection, plaque coverage is a critical issue for CAS success; although CEA could remove almost the entire plaque, carotid stents have a free area between the struts where plaque prolapse could happen, with a risk of late embolization (3). It is well known that the free area surface varies according to stent design, with significant differences between open- and closed-cell stents; the latter has better plaque coverage and a lower incidence of plaque prolapse (4). Since the ICSS study was performed, new mesh-covered stents with a small, free-cell area have become available, which potentially could reduce late embolization from the stent-covered plaque. A more aggressive medical therapy in this subset of patients is advisable, but carotid stent characteristics should be taken into account when considering the global risk of late events.

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REPLY: Stent Type and Risk of Late Cerebral Events After Carotid Artery Stenting

Drs. Pacchioni, Ribichini, and Reimers raise an important issue about the effects of stent design on outcomes after carotid artery stenting. The stent design used was reported for 119 of 124 patients who underwent stenting for symptomatic carotid stenosis in the ICSS-MRI (International Carotid Stenting Study-Magnetic Resonance Imaging) substudy; 74 patients (62%) received an open-cell stent